

CASE REPORT

Congenital Falciparum Malaria with Chloroquine Resistance Type II

by

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Abstract

A case of congenital falciparum malaria has been reported. The diagnosis was based on history of illness, clinical manifestations, age of the child and presence of the ring form falciparum malaria in the peripheral blood films.

Treatment with chloroquine showed resistance type II, though treatment with quinine was successful.

In malaria endemic areas, although rare, one must be aware of the possibility of congenital malaria.

Introduction

Congenital malaria is rare but well documented infectious disease. In the past 50 years there had only been three reported cases of congenital malaria in the United States (McQuay et al., 1967; Harvey et al., 1969). A case of congenital malaria occurring in England was recorded in 1924 by Jones and Brown (cited from Tanner et al., 1935). In Gambia, there is a high incidence of malaria. Logie et al. (1973) did not find any cases of congenital malaria among infants born to 234 mothers in spite of a parasitemia rate of 28% among the mothers and a 32% positive placental blood. In similar studies by Cannon in 1958, there was no case of congenital malaria among 117 newborns, 25.6% of

whom though were found to have an infected placenta.

The transplacental route of infection in malaria is beyond dispute. It has been substantiated by over 150 reported cases of congenital malaria (Thompson et al., 1977). Congenital infection may be proved in two ways, either by finding plasmodium in the blood of the child immediately after birth, or by demonstrating it in the blood of the offspring of malarious mothers born in non-malarial districts (Tanner et al., 1935).

The following is a case of congenital malaria in a baby, who was born in Sawangan village, Minahasa, well known for its malaria endemicity.

Case Report

D.K., a five weeks old Minahasa girl from Sawangan village, was admitted to the Tropical Disease subdivision, Department of Child Health, Faculty of Medicine/Gunung Wenang Hospital, Manado on January 28, 1988. The main complaints were fever and pallor. She had fever on and off 3 days before admission. There were neither chills nor convulsions.

When she was one week old, she had had fever also. An attending physician had found hepatosplenomegaly and a blood peripheral film showed rings of falciparum malaria. The physician at the Pediatric OPD Gunung Wenang Hospital had advised a hospital admission which was refused then. The fever dropped after one day medication and the drug was stopped since then.

The pallor was noticed one day before admission. She was a home delivery, attended by a midwife. Her mother had had fever and chills one week before delivery. She was the first child.

Physical examination on admission revealed a pale, fully alert girl. The body weight was 3.8 kg and the body length 54 cm. The pulse rate was 120/minute, the respiration rate 36/minute and temperature 38.5° C. The heart and lungs were normal. The liver was palpable 4 cm below the costal margin and the spleen enlarged to the size of Schuffner V.

Laboratory investigation on admission revealed the following: Hb. 6.6 g/dl.,

leucocytes 7.700/mm³, platelets 90.000/mm³, reticulocytes 2.5% and the differential count basophils 0%, eosinophils 0%, stab 1%, segment 22%, lymphocytes 76% and monocytes 1%. The blood was positive for falciparum malaria but the G6PD level was normal. Peripheral blood film showed normochrom and normocytic erythrocytes with several spherocytes; the platelets count were less than normal.

The diagnosis was congenital malaria and anemia. She was treated with chloroquine 1 × 10 mg/kgBW/day for 3 consecutive days and a blood transfusion.

With blood transfusion the hemoglobin rose to 9.5 g/dl. on the next day. But on the fifth day the hemoglobin dropped to 7.6 g/dl thus needing a second transfusion.

On February 4, 1988, seven days after the course of chloroquine, the peripheral blood film was still positive for malarial parasites. The child was then treated with quinine 30 mg/kgVW/day in three divided doses for 7 days. The parasites were negative in the next serial peripheral blood film examination.

On February 12, 1988 laboratory examination revealed haemoglobin level of 9.5 g/dl and malaria parasites were negative. The patient was discharged in a good condition. One month later the patient came to Pediatric OPD for DTP and polio vaccination. Her body weight was 5.4 kg and the child was healthy.

Discussion

Malaria is an uncommon disease in the neonatal period and early infancy because of the transplacental barrier, the presence of an acquired immunity from the mother especially in endemic malarious regions (Thapa et al., 1987).

Humoral immunity plays an important

role in the host defence mechanism against malaria (Butcher et al., 1973) and therefore the exacerbations of infection during pregnancy could be due to a reduction of antibody production particularly the immunoglobulin G.

There is also a significant reduction of

the indirect fluorescent antibody (IFA) titre in the third trimester of pregnancy when compared to the non-pregnant control (Campbell et al., 1980). IFA is considered to be a specific antimalarial antibody. The criteria of congenital malaria are either parasites observed in the infant within 7 days after birth or by epidemiological reasoning, excluding the transmission (Loke, 1982).

In this case, the malaria was observed when the child was one week of age. Thus infection by mosquito bite seemed to be almost impossible since the incubation period of falciparum malaria is 9-16 days (Mashaal, 1986).

The classification of congenital malaria (Mashaal, 1986) is as follows:

(1) True congenital malaria (acquired during pregnancy)

The unborn child acquired malaria from his mother owing to failure of the barrier action of the placenta. The parasites are found in the peripheral blood of the baby within 48 hours after birth. This is due to the pathological process or the discontinuity of the placental syncytium for one or other reason. The newborn reveals symptoms at birth or at the first two days.

(2) False congenital malaria (acquired during labor)

The majority of reported congenital malaria cases fall under this category. This occurs in premature separation of the placenta or when labor is prolonged. In a study by Marshall in the Solomon Islands (1983), 11 women in Malaita had parasitemia and 10 of their placentae were infected. The newborn may reveal symptoms for the first time after 3 to 5 weeks or more after birth.

We believed that this case was a true congenital malaria because the symptoms and

signs as well as parasites were found at 1 week of age. We might have diagnosed it earlier if the child had come sooner after birth. The mechanism of congenital malaria had been investigated in several studies using radioactive-labelled erythrocytes injected to the mother, which can then be detected in the fetal blood after delivery (Loke, 1982). Recently, investigators have looked for the presence of Rh⁺ cells in the blood of Rh⁻ infants born to Rh⁺ mother as an indicator for the maternal erythrocytes. Using a fluorescent antibody technique, Jennings and Clauss in 1978 detected Rh⁺ cells in only two out of 105 (1.9%) cases and therefore concluded that the maternofetal transfer of erythrocytes is rare.

In congenital malaria, the following signs and symptoms are detected (Mashaal, 1986): (a) mild temperature elevation; (b) anorexia and irritability in some cases; (c) hepatosplenomegaly, a frequent finding; (d) pallor and jaundice, frequent; (e) hemolytic anemia, it may occur particularly in *P. falciparum*; (g) thrombocytopenia; and (h) gastrointestinal manifestations: vomiting, diarrhea, and dehydration.

In this case, we found most of the signs and symptoms as mentioned above, such as: mild temperature elevation, hepatosplenomegaly, pallor, hemolytic anemia and thrombocytopenia.

In congenital malaria, treatment with primaquine is not necessary because congenital malaria is a form of transfusion malaria and has no exoerythrocyte (liver) stage.

The child was treated with chloroquine (Nivaquine^R) 10 mg/kgBW/day for 3 days. Seven days after the treatment, the malarial parasites decreased from positive three to positive one, indicating resistance to chloroquine type II (WHO, 1980). In Manado, during 1981 to 1985, 6 of 21

(28,6%) malaria children were resistant II to chloroquine (Rampengan and Rampengan, 1986). On the 8th day the child was treated with quinine 30 mg/kgBW/day for 7 days and the parasites were no more found in the next serial peripheral blood film examination.

During hospital stay, the child needed a second transfusion because the haemoglobin level dropped to 7.6 g/dl., as we considered it to be lower than 7.6 g/dl. a second transfusion was performed.

After 15 days of hospitalization, the patient was discharged in a good condition.

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